Page 47, line 12:

In another embodiment of the invention, the altered or mutated forms of structure (I) are obtained by substituting at least one amino acid with a non-conservative amino acid. Those of skill in the art will recognize that such substitutions should not substantially alter the amphipathic and/or structural properties of the helix discussed, supra. Thus, in certain instances it may be desirable to substitute one or more pairs of amino acids so as to preserve the net properties of the helix. Further guidance for selecting appropriate amino acid substitutions is provided by the peptide sequences listed in ~~TABLE X~~ TABLE IX (see, Section 8.3, infra).

Page 64, line 10:

In still another preferred embodiment, the ApoA-I agonists are selected from the group of peptides set forth below:

peptide **191**	PVLDLLRELLEELKQKLK*	(SEQ ID NO:191);
peptide **192**	PVLDLFKELLEELKQKLK*	(SEQ ID NO:192);
peptide **193**	PVLDLFRELLEELKQKLK*	(SEQ ID NO:193);
peptide **194**	PVLELFRELLEELKQKLK*	(SEQ ID NO:194);
peptide **195**	PVLELFKELLEELKQKLK*	(SEQ ID NO:195);
peptide **196**	PVLDLFRELLEELKNKLK*	(SEQ ID NO:196);
peptide **197**	PLLDLFRELLEELKQKLK*	(SEQ ID NO:197);
peptide **198**	GVLDLFRELLEELKQKLK*	(SEQ ID NO:198);
peptide **199**	PVLDLFRELWEELKQKLK*	(SEQ ID NO:199);
peptide **200**	NVLDLFRELLEELKQKLK*	(SEQ ID NO:200);
peptide **201**	PLLDLFKELLEELKQKLK*	(SEQ ID NO:201);
peptide **202**	PALELFKDLLEELRQKLR*	(SEQ ID NO:202);
peptide **203**	AVLDLFRELLEELKQKLK*	(SEQ ID NO:203);
peptide **204**	PVLDFFRELLEELKQKLK*	(SEQ ID NO:204);
peptide **205**	PVLDLFREWLEELKQKLK*	(SEQ ID NO:205);
peptide **206**	PLLELLKELLEELKQKLK*	(SEQ ID NO:206);
peptide **207**	PVLELLKELLEELKQKLK*	(SEQ ID NO:207);

peptide **208**	PALELFKDLLEELRQRLK*	(SEQ ID NO:208);
peptide **209**	PVLDLFRELLNELLQKLK	(SEQ ID NO:209);
peptide **210**	PVLDLFRELLEELKQKLK	(SEQ ID NO:210);
peptide **211**	PVLDLFRELLEELOQOLO*	(SEQ ID NO:211);
peptide **212**	PVLDLFOELLEELOQOLK*	(SEQ ID NO:212);
peptide **213**	PALELFKDLLEEFRQRLK*	(SEQ ID NO:213);
peptide **214**	pVLDLFRELLEELKQKLK*	(SEQ ID NO:214);
peptide **215**	PVLDLFRELLEEWKQKLK*	(SEQ ID NO:215);
~~peptide **229**~~	PVLELFERLLEDLQKKLK	~~(SEQ ID NO:229);~~
peptide **230**		(SEQ ID NO: 230);
~~peptide 230~~	PVLDLFRELLEKLEQKLK	~~(SEQ ID NO:230);~~
peptide **231**		(SEQ ID NO:231);
~~peptide **231**~~	PLLELFKELLEELKQKLK*	~~(SEQ ID NO:231);~~
peptide **232**		(SEQ ID NO:232);

in either the N- and/or C-terminal blocked or unblocked forms.

Page 66, line 34:

In a final preferred embodiment, the ApoA-I agonists are not any of the peptides listed in ~~TABLE X~~ TABLE IX (Section 8.3, infra) which exhibit an LCAT activation activity of less than 38% as compared with native human ApoA-I.

Page 85, line 9:

6. EXAMPLE: SYNTHESIS OF PEPTIDE AGONISTS OF ApoA-I

The peptides described in ~~TABLE X~~ TABLE IX (Section 8.3, infra) were synthesized and characterized as described in the subsections below. The peptides were also analyzed structurally and functionally as described in Sections 7 and 8, infra.

Page 87, line 15:

6.2 SYNTHESIS OF PEPTIDE AMIDES

Where indicated in ~~TABLE X~~ TABLE IX (Section 8.3, infra), peptide amides were

synthesized using a Rink amide resin containing the Fmoc-Rink amide handle 4-(2′, 4′-dimethylphenyl)-Fmoc-phenoxymethyl (Rink, 1987, Tetrahedron Lett. 28: 3787-3790) and the synthesis protocols described in section 6.1, supra.

6.3 SYNTHESIS OF N-TERMINAL ACYLATED PEPTIDES

Where indicated in ~~TABLE X~~ TABLE IX (section 8.3, infra), N-terminal acylated forms of the peptides were prepared by exposing the resin-bound peptide prepared as described in section 6.1 or 6.2, supra, to an appropriate acylating agent.

Page 91, line 13:

After background ~~substraction,~~ subtraction, spectra were converted to molar ellipticity (θ) per residue in deg. cm^{-2} $dmol^{-1}$. The peptide concentration was determined by amino acid analysis and also by absorption spectrometry on a Perkin Elmer Lambda 17 UV/Visible spectrophotometer when the peptide contained a chromophore (~~tryptophane,~~ tryptophan, dansyl, ~~naphtylalanine~~ naphthylalanine).

Page 92, line 29:

7.1.3 RESULTS

The degree of helicity (%) of the free, unbound peptides (free), the peptide-SUV complexes (SUVs), the peptide-micelle complexes (mics) and the peptide-TFE solution (TFE) are reported in ~~TABLE X,~~ TABLE IX, section 8.3, infra.

Page 93, line 7:

Referring to ~~TABLE X,~~ TABLE IX, section 8.3, infra, it can be seen that those peptides which exhibit a high degree of LCAT activation ($\geq$38%) generally possess significant α-helical structure in the presence of lipids ($\geq$60% helical structure in the case of unblocked peptides containing 22 or more amino acids or blocked peptides containing 18 or fewer amino acids; $\geq$40% helical structure in the case of unblocked peptides containing 18 or fewer amino acids), whereas peptides which exhibit little or no LCAT activation possess little α-helical structure. However, in some instances, peptides which contain significant α-helical structure in the presence of lipids do not exhibit significant LCAT activation. As a consequence, the ability of the core peptides of the invention to adopt an α-helical structure in

the presence of lipids is considered a critical feature of the core peptides of the invention, as the ability to form an α-helix in the presence of lipids appears to be a prerequisite for LCAT activation.

7.2 FLUORESCENCE SPECTROSCOPY

The lipid binding properties of the peptides synthesized in Section 6, *supra*, were tested by fluorescence measurements with labeled peptides, in the present case ~~Tryptophane~~ tryptophan (Trp or W) or ~~Naphtylalanine~~ naphthylalanine (Nal). The fluorescence spectra were recorded on a Fluoromax from Spex (Jobin-Yvon) equipped with a Xenon lamp of 150W, two monochromators (excitation and emission), a photomultiplier R-928 for detection sensitive in the red up to 850 nm and a thermoelectric magnetic stirred cell holder. Quartz Suprasil cuvettes were used for measurements in the micromolar concentration range. A device of variable slits (from 0.4 to 5 nm) allows modulation of the incident and emitted intensities according to the concentration of peptide used. The reported values are in general the average of between 2 to 4 spectra. The peptide concentration is determined by absorption spectrometry on a Philips PU 8800 using the absorption band of the Trp ($\epsilon_{280\,nm} = 5{,}550$ $M^{-1}cm^{-1}$ in Tris buffer) or the Nal ($\epsilon_{224\,nm} = 92{,}770$ $M^{-1}cm^{-1}$ in methanol).

Page 94, line 35:

The fluorescence spectra of the peptides were characterized by the wavelength at their maximum of fluorescence emission and by their quantum yield compared to NATA in the case of peptides labeled with a ~~tryptophane~~ tryptophan. The process of binding to lipids was analyzed by calculating the shift of the wavelength at the maximum of fluorescence emission, (λ_{max}), and the variation of the relative fluorescence intensity of emission versus the lipid concentration. The relative fluorescence intensity is defined as the following ratio: $(I - I_0)_{\lambda max}/I_{0\lambda max}$. I and I_0 are both measured at the (λ_{max}) corresponding to the initial free state of the peptide, i.e., without lipids. I is the intensity at a defined lipid to peptide ratio and I_0 is the same parameter measured in absence of lipids. The absence of these variations is relevant ~~of~~ to the absence of interactions of the peptides with the lipids.

Page 95, line 36:

In buffer at a concentration of 2 µm, the maximum of the ~~tryptophane~~ tryptophan fluorescence emission (λ_{max}) of peptide **199** (SEQ ID NO:199) is 348 nm. This corresponds

to a ~~tryptophane~~ tryptophan which is relatively exposed to the aqueous environment when compared to NATA (λ_{max} = 350 nm). Peptide **199** (SEQ ID NO:199) binds very effectively to EPC/Chol (20:1) small unilamellar vesicles as demonstrated by the burying of the ~~tryptophane~~ tryptophan (the wavelength for the [tryptophane] tryptophan maximum fluorescence emission shifts from 348 nm to 325 nm) and the high fluorescence intensity exaltation (See Table VII). The burying of the ~~tryptophane~~ tryptophan residue is maximal for a lipid to peptide molar ratio of about 100.

Page 100, line 16:

8.2 PURIFICATION OF LCAT

For the LCAT purification, dextran sulfate/Mg^{2+} treatment of human plasma is used to obtain lipoprotein deficient serum (LPDS), which is sequentially chromatographed on Phenylsepharose, Affigelblue, ConcanavalinA sepharose and anti-ApoA-I affinity chromatography, as summarized for a representative purification in ~~TABLE IX,~~ TABLE VIII, below:

Page 101, line 1:

~~TABLE IX~~ TABLE VIII

Page 103, line 35:

8.3 RESULTS

The results of the LCAT activation assay are presented in ~~TABLE X,~~ TABLE IX, infra.

Page 104, line 1:

~~TABLE X~~ TABLE IX

Page 119, line 1:

In ~~TABLE X,~~ TABLE IX, * indicates peptides that are N-terminal acetylated and C-terminal amidated; ' indicates peptides that are N-terminal dansylated; sp indicates peptides that exhibited solubility problems under the experimental conditions; X is Aib; Z is Nal; O is Orn; He (%) designates percent helicity; mics designates micelles; and ~ indicates deleted amino acids.